CLINICAL PROFILE OF

STROKE

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CERTIFICATE

This is to certify that the dissertation titled **"CLINICAL PROFILE OF STROKE"** is the bonafide original work of DR. S.A.K.NOOR MOHAMED in partial fulfillment of the requirements for M.D. Branch – I (General Medicine) Examination of the Tamilnadu DR. M.G.R Medical University to be held in SEPTEMPER 2006. The Period of study was from septemper 2005 to February2006.

PROF. S. NATARAJAN, M.D. Professor and Head of the Dept. of Medicine, Govt. Stanley Medical College and Hospital Chennai-600 001.	**PROF. A.K.GEETHADEVI,M.D.** Addl. Professor of Medicine Govt. Stanley Medical College and Hospital Chennai-600 001.

DEAN
Govt. Stanley Medical College & Hospital,
Chennai – 600 001.

DECLARATION

I, **DR. S.A.K.NOOR MOHAMED,** solemnly declare that dissertation titled **" CLINICAL PROFILE OF STROKE "** is a bonafide work done by me at Govt.StanleyMedicalCollegeandHospital from septemper2005 to February2006 underguidance and supervision of my unit chief **PROF. A.K.GEETHADEVI,M.D.** Addl. Professor of Medicine.

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(DR.S.A.K.NOORMOHAMED)

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CONTENTS

CLINICAL PROFILE OF

STROKE

1. INTRODUCTION

Stroke is an emerging epidemic and is the third leading cause of death. The term stroke encompasses [1]

Cerebral infarction

Intra cerebral hemorrhage

Cerebral venous thrombosis

Subarachnoid hemorrhage

Based on a retrospective analyses of subjects admitted in urban hospitals in India it was found that stroke constitutes nearly 2% of all hospital cases and 20% of neurological admissions.[2] The incidence in younger population (below 40 years) is high (13 to 32%) when compared to European countries

A Random survey on 2,58,576 residents in urban areas of Vellore revealed only 147 hemiplegic subjects presumed to be Vascular origin. Thus the prevalence rate for hemiplegia in south India was reported to be 56.9 per 1,00,000 as compared 150 to 186 per 1,00,000 for USA and Europe[3]

2. AIM OF THE STUDY

To study 200 cases of stroke with complete analysis of Age, Sex, Incidence, Types of stroke, other risk factors, Clinical presentation, Onset, Associated features, and CT scan findings

3.REVIEW OF LITERATURE

DEFINITION

A stroke(cerebrovascularaccident) is a rapidly developing episode of focal and at times global loss of cerebral function with symptoms lasting more than 24hours or leading to death with no apparent cause other than that of vascular origin [4]

.

Transient ischaemic attack (TIA) is an acute loss of focal cerebral or monocular function with symptoms lasting less than 24hours.

Reversible ischaemic neurological deficit(RIND) refers to neurological deficit that disappears within 2-3 days of onset

EPIDEMIOLOGY

RISK FACTORS [5]

NON-MODIFIABLE RISK FACTORS

Age : Increasing age is most powerful riskfactor for cerebral infarction , intracerebralhemorrhage and subarachnoidhemorrhage as well as TIA

Sex : men are more at risk for ischaemicstroke than woman upto 75 years of age.

Race : the risk is more in Blacks than Whites

Family history : there is an increased incidence of stroke individuals who have a first degree relative affected with stroke or who havepaternal or maternal history of death or disability due to stroke

MODIFIABLE RISK FACTORS [5]

Hypertension : it is an important predisposing factor not only for cerebral hemorrhage but also for infarction. Both systolic and diastolic pressures contribute to the risk though there is no critical level above which it operates The reduction of BP by 10-12mmHg systolic and 5-6mmHg diastolic was found to be associated with 38% reduction in stroke incidence.

Diabetes mellitus : it is an independent risk factor for stroke. It increases the susceptibility to coronary , femoral , and cerebral atherosclerosis. The relative risk increases two to fourfold in diabetics patients with diabetic complications like retinopathy and autonomic neuropathy have a higher incidence of ischemic stroke.

Hypercholesterolaemia : it is a secondary risk factor indirectly

affecting the risk of stroke. Reducing high cholesterol levels decreases the incidence of coronary artery disease which is the main cause mortality in patients with cerebrovascular disease.

Nonvalvular atrial fibrillation : is common in the age group of 65 – 85 years attributes to a five fold increase in the incidence of embolic stroke especially in those patients who have recent CCF , arterial hypertension and prior thromboembolism

Cigarette smoking : increases ischemic stroke in men and women by predisposing to carotid atherosclerosis. Smokers experience a reduction in stroke risk only after 5 years following cessation of smoking.

Alcohol : light to moderate use reduces the risk by increasing the HDL concentration, where as heavy drinking increases the risk more than on alcoholic drink per day for women and more than two per day men raises the BP causes obesity and triglycerides levels thereby increasing the risk.

TIA : three times greater risk for subsequent stroke or death due to vascular causes ,especially those with hemispherical TIAs.patients with a previous history of stroke are at risk for a subsequent stroke. The risk of stroke recurrence is increased by the presence of dementia

Carotid artery stenosis : Asymptamatic Carotid artery stenosis less than 75% carries a risk of 1.3% annually where risk increases to 10.5% per year if the stenosis is greater than 75%. Ulcerated .echoluscent and heterogenous

plaques with a softcore are at higher risk for embolism.

Blood factors : Elevated haematocrit , haemoglobin and viscosity of the blood is associated with increased risk of ischemic stroke. High mean levels of plasma fibrinogen, factor VIII , von willebrand's factor, anti-thrombin III and lower mean levels of protein C is associated with increased risk The antiphospholipid antibodies is associated with increased risk of ischemic stroke Serum folate concentration less than or equal to 9.2 nmol/L alone may be risk factor ischemic stroke

Hormones : high dose estrogen oral contraceptives increase the risk of stroke in young women , while postmenopausal estrogen supplementation reduces the risk. The risk of cerebral infarction is increased in the 6 weeks following delivery

Others : abdominal or truncal obesity, physical inactivity, habitual snoring are associated with increased risk of ischemic stroke

PATHOPHYSIOLOGY

stroke is 90% ischemic and 10 % hemorrhagic.[6] Abrupt disturption of focal cerebral blood flow causes acute ischemic stroke .The causes of decreased cerebral blood flow include abrupt occlusion of small penetrating arteries arterioles, single or multiple arterial stenoses, arteritis, arterial dissection, venous occlusion, and profound anaemia.

When cerebral blood flow falls below a critical value of 20ml /100g/min there is a loss of neuronal electrical function which is a reversible stage.when cerebral blood flow falls below a critical value of 10ml / 100g/min then aerobic mitochondrial metabolism fails and anaerobic metabolism leads to lactic acidosis.

As a sequel to this sodium and water enters the cell and potassium leaks out of the cell due to failure of energy dependent intracellular homoeostasis leading to irreversible cell death.

Based on these facts the concept of ischemic penumbra was formulated. It is an area of brain that has reached the reversible stage of electrical failure but has not yet passed into the irreversible stage. Thrombolytic agents are used in this time window to salvage the ischemic penumbra zone.

Common causes

Thrombosis

Lacunar stroke (small vessel)

Large vessel thrombosis

Dehydration

Embolic occlusion

Artery-to-artery

Carotid bifurcation

Aortic arch

Arterial dissection

Cardioembolic

Atrial fibrillation

Mural thrombus

Myocardial infarction

Dilated cardiomyopathy

Valvular lesions

Mitral stenosis

Mechanical valve

Bacterial endocarditis

Paradoxical embolus

Atrial septal defect

Patent foramen ovale

Atrial septal aneurysm

Spontaneous echo contrast

Uncommon causes

Hypercoagulable disorders

Protein C deficiency

Protein S deficiency

Antithrombin III deficiency

Antiphospholipid syndrome

Factor V Leiden mutation

Prothrombin G20210 mutation

Systemic malignancy

Sickle cell anemia

Beta Thalassemia

Polycythemia vera

Systemic lupus erythematosus

Homocysteinemia

Thrombotic thrombocytopenic purpura

Disseminated intravascular coagulation

Dysproteinemias

Nephrotic syndrome

Inflammatory bowel disease

Oral contraceptives

Venous sinous thrombosis

Fibromuscular dysplasia

Vasculitis

Systemic vasculitis
(PAN, Wegner's, Takayasu's, giant cell arteritis)

Primary CNS vasculitis

Meningitis (syphilis, tuberculosis, fungal, bacterial, zoster)

Cardiogenic

Mitral valve calcification

Atrialmyxoma

Intra cardiac tumor

Marantic endocarditis

Libman-Sacks endocarditis

Subarachnoid hemorrhage vasospasm

Drugs: cocaine, amphetamine

Moyamoya disease

Eclampsia

CLINICAL SYNDROMES [5]

TRANSIENT ISCHAEMIC ATTACKS

TIAs are abrupt in onset , brief in duration and recovery is possible in 24 hours . Recurrent TIAs can occur. Recognition and treatment is important as a complete stroke can be prevented.

Carotid TIAs

These are characterized by monocular blindness with recovery within Few minutes, visual field disturbance in the form of transient hemianopia and speech disturbance due to dominant hemispherical dysfunction. Hemiparesis and hemisensory loss can occur both in vertebro basilar TIAs as well as carotid TIAs.

Vertebrobasilar TIAs

These are characterized by prominent visual symptoms like Diplopia, homonymous hemianopia and cortical blindness, transient vertigo and dizziness, unsteadiness due to cerebellar dysfunction. And transient lower cranial nerve symptoms like dysarthria, perioral numbness, nasal regurgitation.

Drop attacks occur without any warning .It is thought to be due to ischemia of relays in reticular systems which normally function as part of reflex antigravity mechanism.

Subclavian steal

This syndrome occurs when there is occlusion of the Subclavian artery proximal to origin of vertebral artery which results in retrograde flow of blood down the vertebral artery during exercising the arm thereby leading on to symptoms of hindbrain ischaemia.

Ministrokes

Ministrokes are characterized by episodic ischaemic symptoms which recover within 24 hours but there are small infarcts or hemorrhage in CT. They are as significant as the TIAs

Stroke in evolution

Stroke in evolution refers to the slow progression of neurological deficit over several hours.

Major strokes

Major stroke are of sudden onset with loss of consciousness at the onset or soon after it is very difficult to distinguish clinically between infarction or hemorrhage. Headache and vomiting if present, usually denotes hemorrhage if associated with rapid loss of consciousness. Atherothromboembolism is suggested by the presence of bruit over the carotid arteries.

Unusual types of stroke

Multi-infarct dementia

Watershed infarction

Classification of stroke on the basis of oxfordshire community stroke

Sub classification [7]

Total anterior circulation syndrome (TACS)

Implies a large cortical stroke in middle cerebral or middle and anterior cerebral artery territories.it is characterized by a combination of

- new higher cerebral dysfunction
- Homonymous visual field defect
- an ipsilateral motor and / or sensory deficit involving at least two out of three areas of the face , arm or leg

Partial anterior circulation syndrome

Implies a cortical stroke in middle or anterior cerebral arterial

territory . They are patients with two out of three components of the Total anterior circulation syndromes or new higher cerebral dysfunction alone or motor / sensory deficit more restricted than those classified as a TACS .

Lacunar syndrome

Implies a subcortical stroke due to a small vessel disease

- Pure motor stroke
- Pure sensory stroke
- sensory motor stroke
- ataxic hemipaesis

evidence of higher cortical dysfunction or disturbance of consciousnes excludes Lacunar syndrome

posterior circulation syndrome

ipsilateral cranial nerve palsy with contralateral motor and sensory deficit bilateral motor and / or sensory deficit disorder of conjugate eye movement cerebellar dysfunction without ipsilateral long tract involvement isolated homonymous visual field defects

ANTERIOR CEREBRAL CIRCULATION SYNDROME

no	Signs and symptoms	Structures involved
1.	Paralysis of opposite foot and leg	Motor leg area
2.	Lesser degree of paresis of opposite arm	Involvement of cortical / corona radiata of arm fibres
3.	Cortical sensory loss over toes, foot and leg	Sensory area for foot and leg
4.	Urinary incontinence	Sensory motor area in para central lobule
5.	Contralateral grasp reflex,suckling reflex, gegenhalten	Medial surface of the posterior frontal lobe
6.	Abulia,slowness,delay,intermittentinterruption, lackofspontaneouslywhispering,reflex distraction to sights and sounds	Cingulatedgyrus,medial inferiorportion of frontal, parietal and temporal lobes
7.	Impairement of gait and stance (gait apraxia)	Frontal cortex near leg motor area
8.	Dyspraxia of left limbs, Tactile aphasia in left limbs	Corpus callosum

MIDDLE CEREBRAL CIRCULATION SYNDROME

no	Signs and symptoms	Structures involved
1	Paralysis of the contralateral face, arm, and leg; sensory impairment over the same area (pinprick, cotton touch, vibration, position, two-point discrimination, stereognosis, tactile localization, barognosis, cutaneographia)	Somatic motor area for face and arm and the fibers descending from the leg area to enter the corona radiata and corresponding somatic sensory system
2	Motor aphasia	Motor speech area of the dominant hemisphere
3	Central aphasia, word deafness, anomia, jargon speech, sensory agraphia, acalculia, alexia, finger agnosia, right-left confusion (the last four comprise the Gerstmann syndrome	Central, suprasylvian speech area and parietooccipital cortex of the dominant hemisphere
4	Conduction aphasia	Central speech area (parietal operculum)
5	Apractognosia of the minor hemisphere (amorphosynthesis), anosognosia, hemiasomatognosia, unilateral neglect, agnosia for the left half of external space, dressing "apraxia," constructional "apraxia," distortion of visual coordinates, inaccurate localization in the half field, impaired ability to judge distance, upside-down reading, visual illusions (e.g., it may appear that another person walks through a table)	Nondominant parietal lobe (area corresponding to speech area in dominant hemisphere); loss of topographic memory is usually due to a nondominant lesion, occasionally to a dominant one
6	Homonymous hemianopia (often homonymous inferior quadrantanopia)	Optic radiation deep to second temporal convolution
7	Paralysis of conjugate gaze to the opposite side	Frontal contraversive field or projecting fibers

POSTERIOR CEREBRAL CIRCULATION SYNDROME

Peripheral territory

no	Signs and symptoms	Structures involved
1	Homonymous hemianopia (often upper quadrantic)	Calcarine cortex or optic radiation
2	Bilateral homonymous hemianopia, cortical blindness, awareness or denial of blindness; tactile naming, achromatopia (color blindness), failure to see to-and-fro movements, inability to perceive objects not centrally located, apraxia of ocular movements, inability to count or enumerate objects, tendency to run into things that the patient sees and tries to avoid	Bilateral occipital lobe with possibly the parietal lobe involved
4	Verbal dyslexia without agraphia, color anomia	Dominant calcarine lesion and posterior part of corpus callosum.
5	Memory defect	Hippocampal lesion bilaterally or on the dominant side only
6	Topographic disorientation and prosopagnosia	nondominant, calcarine, and lingual gyrus.
7	Simultagnosia, hemivisual neglect	Dominant visual cortex, contralateral hemisphere
8	Unformed visual hallucinations, peduncular hallucinosis, metamorphopsia, teleopsia, illusory visual spread, palinopsia, distortion of outlines, central photophobia	Calcarine cortex.
9	Complex hallucinations	nondominant hemisphere.

POSTERIOR CEREBRAL CIRCULATION SYNDROME

Central territory

no	Signs and symptoms	Structures involved
1	Thalamic syndrome: sensory loss (all modalities), spontaneous pain and dysesthesias, choreoathetosis, intention tremor, spasms of hand, mild hemiparesis	Posteroventral nucleus of thalamus; involvement of the adjacent subthalamus body or its afferent tracts
2	Thalamoperforate syndrome: crossed cerebellar ataxia with ipsilateral third nerve palsy (Claude's syndrome)	Dentatothalamic tract and issuing third nerve
3	Weber's syndrome: third nerve palsy and contralateral hemiplegia	Third nerve and cerebral peduncle
4	Contralateral hemiplegia	Cerebral peduncle
5	Paralysis or paresis of vertical eye movement, skew deviation, sluggish pupillary responses to light, slight miosis and ptosis (retraction nystagmus and "tucking" of the eyelids may be associated)	Supranuclear fibers to third nerve, interstitial nucleus of Cajal, nucleus of Darkschewitsch, and posterior commissure
6	Contralateral rhythmic, ataxic action tremor; rhythmic postural or "holding" tremor (rubral tremor)	Dentatothalamic tract

DIAGNOSTIC EVALUATION OF ISCHAEMIC STROKE [8]

The diagnostic evaluation should include parallel assessment of the following.

1. Imaging of the infarct
2. vascular studies
3. cardiac evaluation
4. Haematological and other blood testing

Imaging

CT scan brain is done to differentiate hemorrhage from infarction Investigations must be ordered based on the possible etiology.Cerebral arteriography is needed if intra arterial thrombolysis is contemplated. Angiogram is done if vascular stenosis is suspected if it is negative or if Posterior circulation is suspected then ,TransCranial Doppler is done.

CT versus MRI

1. only a minority infarction demonstrated within 24 hours on CT.

   MRIdocument infarct as early 6 hours
2. anatomic extent and vascular distribution are better delineated in MRI.

   Small infarctions are easily seen.
3. posterior fossa infarctions are better visualized in MRI

CT scan changes in cerebral infarction [9]

Hyper acute infarct < 12 hours

- normal (50 – 60%)
- Hyper dense artery(25 – 50%)
- Obscuration of lentiform nuclei

Acute infarct 12 to 24hours

- Low density basal ganglia
- Loss of grey white interfaces
- Sulcal effacememt

1 to 3days

- Increasing mass effect
- Wedge shaped low density area that involves both grey and Whitematter
- Hemorrhagic transformation may occur(basalganglia and cortex are common sites)

4 to 7days

- Gyral enhacement
- mass effect, oedema persist

1 to 8 weeks

- Contrast enhacement persists

- mass effect resolves

months to years

- encephalomalacicchanges
- volume loss
- calcification rare

Neurological Pearls In Prognosis

predictors of stroke outcome [10]

factors predicting poor outcome

1. age : more than 75 years
2. males : due to lack oestrogen protective effect
3. risk factors : atrial fibrillation , DM , previous stroke
4. clinical findings
    - decreased consciousness at the onset
    - Presence of gaze deviation
    - Headache , nausea ,vomiting in first 24 hours
    - Elevated systolic BP >180 mm Hg on first day
    - Hyperthermia on admission
    - NIHSS score of 16 or more
    - Large vessel disease

5. laboratory findings

- High glutamine in plasma > 200 micromol / L

- CRP concentration > 10.2 mg / l within 72 hours
- Hyperglycaemia > 7mmol / l
- Platelet count < 150000 due to increased bleeding

6.neuro imaging studies

- Hyper density in a major intra cranial artery
- Early CT changes within 6 hours of onset
- >33 % of MCA territory involvement / multiple territory involvement with mass effect

- Hemorrhagic transformation on follow up CT / on intra cranial Doppler persisting MCA occlusion for hours
- No flow on SPECT perfusion patterns
- Carotid artery occlusion on conventional angiogram

- MCA , basilar artery occlusion on angiogram
- MRI – abnormal PWI in diffusion and perfusion weighted imaging
- MRA – absence of MCA is associated with poor prognosis

TREATMENT

The first goal is to prevent or reverse brain injury

The second goal is to obtain an accurate understanding of the stroke mechanism so one can halt progression of brain injury or begin to prevent a second stroke

Treatments designed to reverse or lessen the amount of tissue infarction fall within five categories:

(1) Medical support

(2) Thrombolysis,

(3) Anticoagulation

(4) Antiplatelet agents, and

(5) Neuroprotection.

Medical Support

1. When cerebral infarction occurs, the immediate goal is to optimize cerebral perfusion in the surrounding ischemic area

2 . Preventing the common complications of bedridden patients

infections (pneumonia, urinary tract, and skin) and

deep venous thrombosis with pulmonary embolism.

3. Elevated blood pressure should not be lowered unless there is malignant hypertension or concomitant myocardial ischemia.

If the blood pressure is low, raising it is advisable, using intravenous fluids or vasopressor drugs to enhance perfusion within the ischemic penumbra.

4. treatment of cerebral edema if necessary

Thrombolysis

The use of thrombolytic agents in acute cerebral infarction has been studied extensively.

Angiography performed within a few hours of infarction frequently demonstrates arterial occlusions corresponding to patients presenting signs and symptoms. It is this association of arterial occlusion with acute neurologic symptoms that prompted the study of thrombolytic agents in stroke patients.

Agent used for this purpose is intravenous recombinant tissue plasminogen activator (rtPA)

Indication [11]

Clinical diagnosis of stroke

Onset of symptoms to time of drug administration ≤ 3 h

CT scan showing no hemorrhage or significant edema

Age ≥ 18 years

Consent by patient or surrogate

Administration of rtPA [12]

Intravenous access with two peripheral IV lines (avoid arterial or central placement)

Review eligibility for rtPA

Administer 0.9 mg/kg intravenously (maximum 90 mg) IV as 10% of total dose by bolus, followed by remainder of total dose over 1 h

Continous cuff blood pressure monitoring

No other antithrombotic treatment for 24 h

For decline in neurologic status or uncontrolled blood pressure, stop infusion, give cryoprecipitate, and reimage brain emergently

Avoid urethral catheterization for > 2 h

Contraindication [11]

Sustained BP > 185/110

Platelets < 100,000; HCT< 25%; glucose < 50 or > 400

Use of heparin within 48 h and prolonged PTT, or elevated INR

Rapidly improving symptoms

Prior stroke or head injury within 3 months; prior intracranial hemorrhage

Major surgery in preceding 14 days

Minor stroke symptoms

Gastrointestinal bleeding in preceding 21 days

Recent myocardial infarction

Coma or stupor

Anticoagulation

The role of anticoagulation in atherothrombotic cerebral ischemia is uncertain. Heparinization is generally accomplished by beginning an infusion without bolus and is monitored to maintain the activated PTT at approximately twice normal.

This regimen is maintained for 2 to 5 days. During this time the patient is monitored for hemorrhagic complications, the evaluation is completed decision is made regarding the need for carotid endarterectomy, long-term anticoagulation, or an antiplatelet therapy.

If long-term anticoagulation is chosen, warfarin is administered and heparin discontinued when the international normalized ratio (INR) is in the range of 2 to 3.

Antiplatelet Agents

Aspirin is the only antiplatelet agent that has been prospectively studied for the treatment of acute ischemic stroke. The use of aspirin within 48 h of stroke onset reduced both stroke recurrence risk and mortality minimally

Neuroprotectio

Neuroprotection is the concept of providing a treatment that

prolongs the brain's tolerance to ischemia long enough to allow other measures to be employed to mitigate ischemia.

Hypothermia is probably the most powerful neuroprotectant but is only now the subject of clinical trials.

Primary and Secondary Prevention

Atherosclerosis Risk Factors

Hypertension is the most significant of the risk factors; in general, all hypertension should be treated. coronary artery disease is the most common cause of death in patients with cerebrovascular disease, treatment of hypercholesterolemia seems prudent for both the heart and brain.Tobacco smoking should be discouraged in all patients

Antiplatelet Agents

Platelet antiaggregation agents can prevent atherothrombotic events, including TIA and stroke, by inhibiting the formation of intraarterial platelet aggregates. These can form on diseased arteries, induce thrombus formation, andocclude the artery or embolize into the distal circulation.

Aspirin,

clopidogrel, and

The combination of aspirin plus extended-release dipyridamole are the antiplatelet agents used most for this purpose.

EMBOLIC STROKE

warfarin is more effective than aspirin in preventing ischemic stroke associated with atrial fibrillation.

Anticoagulation Therapy

ATHEROTHROMBOTIC STROKE

There are few data to support the use of long-term warfarin for preventing atherothrombotic stroke, either intracranially or extracranially.

EMBOLIC STROKE

Several recent trials demonstrated that anticoagulation (INR range 2 to 3) in patients with chronic nonvalvular (nonrheumatic) atrial fibrillation prevents cerebral embolism and is safe.

For primary prevention and for patients who have experienced stroke or TIA, anticoagulation with warfarin reduces the risk by about 65% and clearly outweighs the 1% per year rate of major bleeding complication.

Because of the high annual stroke risk in untreated rheumatic heart disease, primary prophylaxis against stroke has not been studied in a double-blind fashion. These patients generally receive long-term anticoagulation.

Anticoagulation also reduces the risk of embolism in acute myocardial infarction. Most clinicians recommend a 3-month course of anticoagulation when there is anterior Q-wave infarction,substantial Left

ventricular dysfunction ,congestive heart failure, muralthrombosis, or atrial fibrillation .Warfarin is recommended long-term if atrial fibrillation persists. Thromboembolism is one of the most serious complications of prosthetic heart valve implantation. Anticoagulation has been proven effective for preventing strokes in this situation, while antiplatelet therapy alone has not.

However, coupled with warfarin anticoagulation, aspirin adds substantial benefit. A greater degree of anticoagulation (INR of 3 to 4, depending on valve type) is recommended for prosthetic heart valve patients.

SURGICAL THERAPY

Surgery for atherosclerotic occlusive disease is largely limited to carotid endarterectomy for plaques located at the origin of the internal carotid artery in the neck

Carotid endarterectomy is a proven effective prophylaxisagainst stroke and TIA.

Stroke Centers and Rehabilitation

Comprehensive stroke units that care for the acute patient followed by rehabilitation services have been shown to improve neurologic outcomes and reduce mortality

Proper rehabilitation of the stroke patient includes early physical, occupational, and speech therapy. It is directed toward educating the

patient and family about the patient's neurologic deficit, preventing the complications of immobility (e.g., pneumonia, deep vein thrombosis and pulmonary embolism,pressure sores of the skin, muscle contractures), and providing encouragement and instruction in overcoming the deficit. The goal of rehabilitation is to return the patient to home and to maximize recovery by providing a safe, progressive regimen suited to the individual patient

INTRA CEREBRAL HEMORRHAGE

Intra cerebral hemorrhage accouts for approximately 15% of the strokes. The overall mortality for this type subtype of stroke is from 25 % to 60% In nearly 70% of patients Hypertension is the commonest cause.

The lipohyalinosis of the small intraparenchymal arteries is the leading cause of hemorrhage.

The microaneurysms of charcot and bouchard is uncertain,but they are found at anatomical sites preferentially affected by ICH.

The non Hypertensive causes include following :

1. bleeding disorders, anticoagulant and fibrinolytic treatment
2. cerebral amyloid angiopathy
3. granulomatous angiitis of the CNS
4. sympathomimetic agents
5. trauma

6. hemorrhagic infarction
7. vascular malformations
8. intra cranial tumours

clinical features:

symptoms of increased ICT

symptoms that are specific for the location of the haematoma

focal neurological deficits

Imaging

The CT scan is sensitive to the high density fresh blood in the parenchyma while MRI can determine the time duration between the hemorrhage and the MRI examination

common sites :

1 . putamen
2 . lobar
3 . thalamus
4 . caudate nucleus
5 . pons
6. cerebellum

Treatment

Control of hypertension

Air way maintenance if the GCS is less than 8

Treatment of coagulation abnormalities

Protamine sulfate is used if the hemorrhage is due to heparin Cryoprecipitate is used if the hemorrhage is due to heparin

Measures for the prevention of further elevation of ICP

Routine anticonvulsants not recommended in patients who do not have seizures at onset due to negligible risk of subsequent epilepsy in them

Patients with lobar hemorrhage and cerebellar hemorrhage can be managed surgically whereas the deep hemorrhages are managed medically.

SUBARACHNOID HEMORRHAGE

Causes

Head trauma,

Rupture of a saccular aneurysm. the most common cause

Bleeding from a vascular anomaly and

Extension into the subarachnoid space from a primary ICH

Idiopathic SAHs .

Clinical Manifestations

Headache

"the worst headache of my life."

sudden headache in the absence of focal neurologic symptoms is the hallmark of aneurysmal rupture

Delayed neurologic deficits

There are four major causes of delayed neurologic deficits;

Rerupture,

Hydrocephalus,

Vasospasm, and

Hyponatremia.

Laboratory Evaluation and Imaging

The hallmark of aneurysmal rupture is blood in the CSF,more than 95% of cases have enough blood to be visualized on a high-quality noncontrast CT scan obtained within 72 h.

If the scan fails to establish the diagnosis of SAH and no mass lesion or obstructive hydrocephalus is found, an LP should be performed to establish thepresence of subarachnoid blood. Lysis of the red blood cells and subsequent conversion of hemoglobin to bilirubin stains the spinal fluid yellow within 6 to 12 h of SAH. This xanthochromic spinal fluid peaks in intensity at 48 h and lasts for1 to 4 weeks, depending on the amount of subarachnoid blood

Four-vessel conventional x-ray angiography (both carotids and both vertebrals) is generally performed to localize and define the anatomic details of the aneurysm and to determine if other unruptured aneurysms exist..

The ECG frequently shows ST-segment and T-wave changes similar to those associated with cardiac ischemia. Prolonged QRS complexincreased QT

interval, and prominent "peaked" or deeply inverted symmetric T waves are usually secondary to the intracranial hemorrhage. Serum electrolytes are obtained because hyponatremia may develop.

Close monitoring (daily or twice daily) of serum sodium is important since hyponatremia can occur precipitously during the first 2 weeks following SAH .

TCD ultrasound assessment of proximal middle, anterior, and posterior cerebral and basilar artery flow is helpful in detecting the onset of vasospasm and in following its course and response to therapy.

TREATMENT

The medical management of SAH centers on airway protection, blood pressure management before and after aneurysm treatment, preventing rebleeding prior to treatment, managing vasospasm, treating hydrocephalus, and treating hyponatremia.

An aneurysm can be "clipped" by a neurosurgeon or "coiled" by a neurointerventional radiologist

Patients who are stuporous should undergo emergent ventriculostomy to prevent cerebral ischemia from high ICP. Medical therapies designed to combat raised ICP (e.g., mild hyperventilation, mannitol, and sedation) can also be used as needed

All patients who are not candidates for early surgical treatment areput on bed rest in a quiet, preferably darkened, room and are given stool softeners to prevent constipation.

Adequate hydration is necessary to avoid a decrease in blood volume predisposing to brain ischemia.

Phenytoin is often given as prophylactic therapy since a seizure may promote rebleeding.

Glucocorticoids may help reduce the head and neck ache caused by the irritative effect of the subarachnoid blood.

Vasospasm remains the leading cause of morbidity and mortality following aneurysmal SAH and treatment of the aneurysm. Treatment with the calcium channel antagonist Nimodipine (60 mg orally q6h) is beneficial.

The most widely accepted therapy for symptomatic cerebral vasospasm is to increase the cerebral perfusion pressure by raising mean arterial pressure through plasma volume expansion and the judicious use of vasopressor agentsusually phenylephrine or dopamine.

Volume expansion helps prevent hypotension, augments cardiac output, and reduces blood viscosity by reducing hematocrit. This method is called "triple-H" (hypertension, hemodilution, and hypervolemic) therapy.

If symptomatic vasospasm persists despite optimal medical

therapy, Intra arterial papaverine and percutaneous transluminal angioplasty are considered.

When chronic hydrocephalus develops, ventricular shunting is the treatment of choice.

Free-water restriction is contraindicated in patients with SAH at risk for vasospasm because hypovolemia and hypotension may occur and precipitate cerebral ischemia.

All patients should have pneumatic compression stockings applied to prevent pulmonary embolism.

CEREBRAL VENOUS THROMBOSIS

Venous sinus thrombosis of the lateral or sagittal sinus or of small cortical veins (cortical vein thrombosis)

Causes

pregnancy and the postpartum period

sepsis

intracranial infections (meningitis)

thrombophilia

polycythemia

sickle cell anemia

proteins C and S deficiency

factor V Leiden mutation (resistance to activated protein C)

antithrombin III deficiency

homocysteinemia

prothrombin G20210 mutation.

oral contraceptives

Clinical features

headache

focal neurologic signs

seizures

signs of increased ICP

coma

Imaging

CT imaging is normal unless an intracranial venous hemorrhage

MR venography or conventional x-ray angiography.

Treatment

Intravenous heparin

Warfarin for 3 to 6 months

Aspirin

4.MATERIALS AND METHODS

1. 200 cases of stroke in government Stanley medical college between september2005 to february 2006
2. Complete history , physical examination were done and clinical diagnosis of the patient was arrived
3. All patients BP , bloodsugar , urea ,serum creatinine , electrolytes , total cholesterol X ray chest , ECG , hemoglobin , total and differential count , and associated Diseases were noted
4. if associated diseases were noted further special work up was done
5 . CT brain plain was done for all patients
6 . contrast enhancement CT brain was done as advised by radiologist
7 . MRI brain was done as advised by neurologist
8 . the results of all the data are expressed in the tabular forms for analysis

Excluded cases

Cerebral tumour

Cerebral abscess

Tuberculoma brain

Old CVA admitted for other diseases

5 . OBSERVATIONS

TABULATIONS

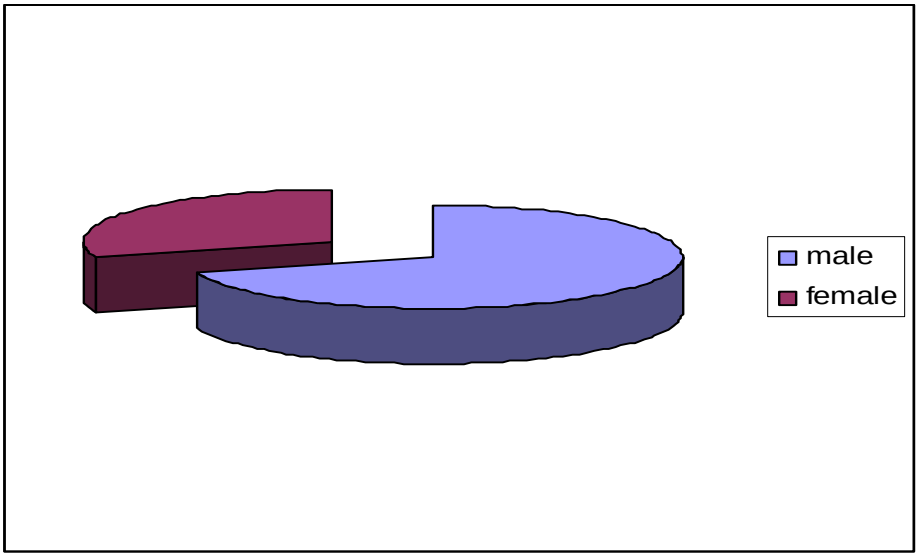
SEX DISTRIBUTION Table (5.1)

Sex	No of cases	%
male	140	70%
female	60	30%

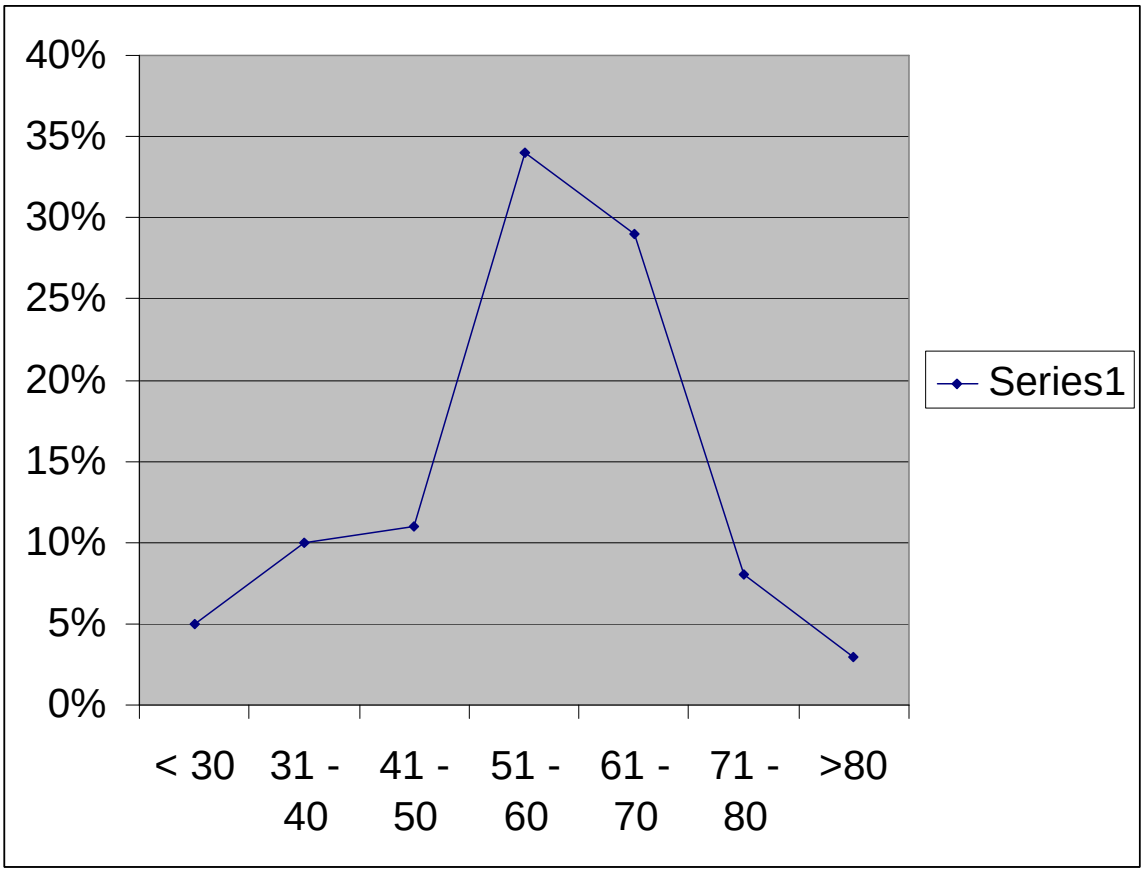
AGE WISE DISTRIBUTION Table (5. 2)

Age	Male	Female	Total	%
< 30	6	4	10	5%
31 - 40	14	6	20	10%
41 - 50	18	4	22	11%
51 - 60	50	18	68	34%
61 - 70	38	20	58	29%
71 - 80	10	6	16	8%
>80	4	2	6	3%
	140	60	6	100%

SEX DISTRIBUTION Fig(5.1)



AGE WISE DISTRIBUTION Fig (5.2)



INCIDENCE OF STROKES (table 5 . 3)

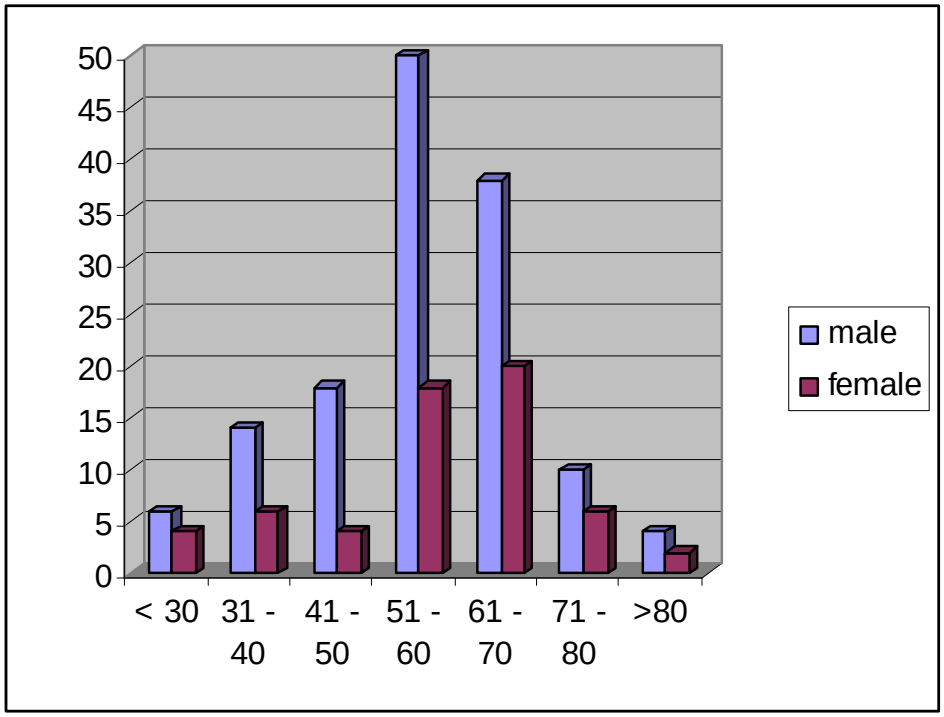
Incidence of strokes	**Male**	**%**	**Female**	**%**	**Total**
Ischemic stroke *	114	57%	48	24%	81%
Intra cerebral hemorrhage	26	13%	10	5%	18%
Cerebralvenousthrombosis	-	-	2	1%	1%
subarachnoid hemorrhage	-	-	-	-	

* cerebral infarction ,TIA, cerebellar infarct

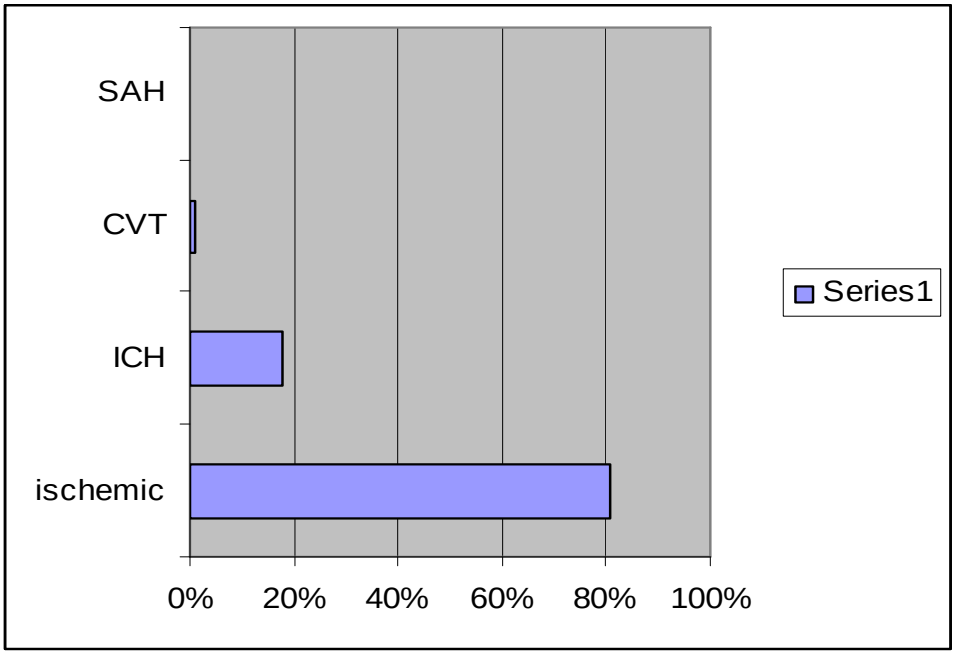
TYPES OF STROKE (table5 . 4)

Types of stroke	**Male**	**%**	**Female**	**%**	**total**
Cerebral infarction	104	52%	40	20%	72%
TIA	8	4%	8	4%	8%
Cerebellar infarct	2	1%	-	-	1%
Intra cerebral hemorrhage	26	13%	10	5%	18%
Cerebral venous thrombosis	-	-	2	1%	1%
subarachnoid hemorrhage	-	-	-	-	-

AGE AND SEX WISE DISTRIBUTION Fig (5.3)



INCIDENCE OF STROKES Fig (5.4)



RISK FACTORS (table 5. 5)

Risk factors	**Male**	**%**	**Female**	**%**	**Total**
hypertension	82	41%	50	25%	66%
diabetes	36	18%	20	10%	28%
Hyper cholesterolemia*	20	17%	8	7%	24%
Non valvular atrialfibrillation	-	-	-	-	-
smoking	72	36%	-	-	36%
alcohol	80	40%	2	1%	41%
TIA	20	10%	8	4%	14%
obesity	4	2%	8	4%	6%

* - for ischemic stroke only

ONSET (table 5 . 6)

Onset	**Male**	**Female**	**Total**
sudden	84	46	65%
gradual	56	14	35%

ACTIVITY OF ONSET (table5 . 7)

Activity of onset	**Thrombosis**	**%**	**Hemorrhage**	**%**
On arising	64	44%	4	12%
Stress	8	6%	6	16%
Activities of daily living	72	50%	26	72%

CLINICAL PRESENTATION (table 5 . 8)

Clinical presentation	Male	%	Female	%	Total
Right hemiplegia	78	39%	26	13%	52%
Left hemiplegia	44	22%	24	12%	34%
Faciobrachial monoplegia	8	4%	2	1%	5%
TIA	8	4%	8	4%	8%
Cerebellar symptoms	2	1%	-	-	1%

ASSOCIATED FEATURES (table 5 . 9)

	Thrombosis	ICH	Total	%
Loss of consciousness	40	12	52	26
Seizures	12	8	20	10
Bladder / bowel involvement	48	8	56	28
Hemianesthesia	6	2	8	4
Headache	56	24	80	40
Hemianopia	4	2	6	3
Facial palsy	136	32	168	84
Aphasia	82	12	94	47

TIA (table 5 . 10)

	Male	**Female**	**Total**	**%**
Weakness / clumsiness	6	4	10	62
Sensory symptoms	2	3	5	31
Dysarthria	2	2	4	25
Dysphasia	3	1	4	25
Unsteadiness	1	-	1	6
Vertigo	1	-	1	6

CARDIAC LESIONS ASSOCIATED WITH STROKE (table 5 . 11)

	Male	**Female**	**Total**
Rheumatic heart diseases	1	3	4
Ischemic heart disease	10	5	15
Mitral valve prolapse	3	3	6
Cardiomyopathies	8	2	10
Aortic sclerosis	6	1	7

ASSOCIATED HISTORY OF OTHER DISEASES (table 5 . 12)

	Male	**Female**	**Total**
Pulmonary tuberculosis	28	12	40
Bronchial asthma	20	8	28
COPD	18	2	20
CRF	6	4	10
Hepatic diseases	2	-	2
Cardiac diseases	28	14	42
Seizures	8	4	12
Psychiatric disorders	2	-	2

CVT(table 5 . 13)

2 Femalecases	**Case No.1**	**Case No. 2**
Age	28	26
Focal neurological signs	+	+
Headache	+	-
Seizures	-	-
papilloedema	+	+

IMAGING (table 5. 14)

	Male	**Female**	**Total**
Cerebral infarction	58	26	84
Cortical atrophy	20	6	26
Multiple infarct	16	4	20
Normal	10	4	14
Cerebellar infarct	2	-	2
Intra cerebral hemorrhage	26	10	36
Cerebral venous thrombosis*	-	2	2

*confirmed by MRI brain

6 . DATA ANALYSIS

SEX DISTRIBUTION (table 5 . 1)

Of the 200 cases studied the males affected were 140 in number while females were 60 cases

The male : female ratio is 2.3 : 1

AGE WISE DISTRIBUTION (table 5 . 2)

young stroke (less than 40 years) in this study is 15 %.the maximum number of stroke occurred between 51 – 60 years is 34 %. The number of cases between 51 – 70 years is 126 out of 200 which is maximum including both sexes (63%). The percentage of cases below 30 and above 80 years is 5% , 3% respectively.

INCIDENCE OF STROKES (table5 . 3)

In this study , Ischemic stroke is 81% , Intra cerebral hemorrhage 18 %, Cerebral venous thrombosis 1 %, subarachnoid hemorrhage 0%

TYPES OF STROKE (table 5 . 4)

Cerebral infarction including embolic stroke constituted 72 %. TIA was 16 cases including 8 males , 8 females patients constituted 8 %.there was 2 male cases. Cerebellar infarct also. Intra cerebral hemorrhage was 18 %.They constituted 36 of 200 cases in both sexes.

Cerebral venous thrombosis also there which was 2 female cases.

No cases of subarachnoid hemorrhage in this study.

RISK FACTORS (table 5 . 5)

Hypertension was the predominant risk factor in about 66 %. In this study HT associated with ischemic stroke was 72%(104 / 144). 28persons out of 36 ICH had HT giving 77%.

In this study DM was present in 28% cases .

In patients with ischemic stroke Hypercholesterolemia was present in 28 out of 114 which constituted 24%.

There was no cases of non valvular atrialfibrillation .

Smoking formed 36% in this study occupied entirely by males

82 of 200 cases were alcohol consumers of which 2 were female patients.

Of these 200 cases 28 patients had TIA before this stroke , 20 males and 8 females.

Obesity was present in 12 cases which 8 were females.

ONSET OF STROKE (table 5 . 6)

Onset of stroke was sudden in 65%

The rest of 35% were gradual onset

ACTIVITY OF ONSET(table 5 . 7)

68 patients developed stroke On arising while 14 persons stroke related to stress. For 98 patients stroke developed while on Activities of daily living.

CLINICAL PRESENTATION (table5 . 8)

86% presented with weakness out of which 52% had Rightside Hemiplegia or hemiparesis, the remaining 34 % on left side. 10 patients presented with Faciobrachial monoplegia. 4% males and 4% females had TIA symptoms. 2 patients had Cerebellar symptoms.

ASSOCIATED FEATURES (table 5 . 9)

In this study Loss of consciousness was present in 52 cases.

Seizures constituted 10%.

Bladder / bowel involvement along with stroke was 56 cases.

Headache was present in 40 %

UMN type Facial palsy involved 84% of stroke patients.

47 % patients were aphasic.

There was symptoms of Hemianesthesia and Hemianopia in 4% and 3% cases respectively.

TIA (table 5 . 10)

Both male and female patients presented with TIA symptoms most Weakness / clumsiness (62%) and other symptoms like Sensory Symptoms (31%). Dysarthria (25%) , Dysphasia (25%), Unsteadiness(6%) Vertigo (6%).

CARDIAC LESIONS ASSOCIATED WITH STROKE(table 5 . 11)

42 patients of 200 had cardiac lesions of these 28 were males and 14

were females . Cardiac lesions predominantly present in males were of Ischemic heart disease, Cardiomyopathies , AorticSclerosis where as females had Rheumatic heart diseases. Mitral valve prolapse was present in both sexes equally.

ASSOCIATED HISTORY OF OTHER DISEASES (table 5. 12)

Among 200 stroke cases , patients had past history of other diseases also. Those were Pulmonary tuberculosis (40cases) , Bronchial asthma (28), COPD(20), CRF(10), Hepatic diseases(2), Cardiac diseases(42), Seizures(12), Psychiatric disorders(2)

CEREBRAL VENOUS THROMBOSIS (table5 . 13)

2 female cases of Cerebral venous thrombosis were present in this study. Both were postpartum by 14 days . One was 28 year old , another was 26. Both cases had focal neurological signs and papilloedema. One had headache. But no seizures.

IMAGING (table 5 . 14)

In this study, among 162 ischemic stroke, 84 patients had Cerebral infarction , Cortical atrophy was present in 26. Multiple infarct was 20 in number whether 14 patients showed normal studies.

7 . DISCUSSION

INCIDENCE

Dalal P.M (1981) from japan studies showed that 4.5% medical admission were from stroke[3]. From this study it was about 5% (200 cases out of 4000 admissions)

SEX

Sacco RL et al study showed males had a greater frequency of stroke than Women [13] . In this study also males affected greater than female.

AGE

In this study the stroke was maximum in the age group between 50 – 70 year about 63%. In western countries the stroke was more in age 75 year [14]

Young stroke (less than 40 years) in this study was 15%. According to Radhakrishnan study (1986) [15] it was 19%

RISK FACTORS

Hypertension was present in 66% of cases is this study. Studies provided by Stroke registry in germany by Arbeitsgruppe schalganfall hessen [16] showed hypertension associated stroke was 72%

In this study 28persons out of 36 ICH had HT giving 77%. According to Queshi AI, Suti MAK, Saflar K et al(1997)[17] HT associated with ICH was 77%

In this study HT associated with ischemic stroke was 72%. Study conducted by Nizam institute of medical sciences [18] Hyderabad showed HTassociated with ischemic stroke was 62%

In this study DM was present in 28% cases; research letters from C.foerch, T.neumann – haefelin et al(1997) [19] showed 27% diabetics among stroke

Hyper cholesterolemia present in this study was 24% among ischemic stroke research letters from C.foerch,T.neumann – haefelin et al(1997) [19] showed hyper cholesterolemia was 23%

Smoking formed 36% in this study. Sandercock et al 1989 [20] showed 27% smoker among stroke patients.

The status of alcohol ingestion as a risk factor for ischemic stroke remains Controversial [21]. Light to moderate regular consumption of alcohol seems to be inversely related to carotid artery and systemic atherosclerosis.[22] Acute &chronic heavy use of alcohol positively correlate with incidence of ischemic stroke

In Honolulu heart study[23], alcohol consumption was associated with Intra cerebral hemorrhage.

In this study previous TIA was 12% among stroke patients. In oxfordshire community project Sandercock et al 1989 [20] showed TIA was 14 %.

ACTIVITY OF ONSET

Thrombotic stroke	In this study	MRSR study [24]
On arising	44%	42%
Stress	6%	2%
Activities of daily living	50%	56%

Intra cerebral hemorrhage	In this study	MRSR study [24]
On arising	12%	18%
Stress	16%	15%
Activities of daily living	72%	67%

SIDE OF LESION

Side of lesion	In this study	Other study [19]
Left	60%	40%
Right	56%	44%

FEATURES OF TIA

Features of TIA	In this study	Other study [25]
Weakness / clumsiness	62%	50%
Sensory symptoms	31%	35%
Dysarthria	25%	23%
Dysphasia	25%	18%
Unsteadiness	6%	12%
Vertigo	6%	5%

CARDIAC LESIONS ASSOCIATED WITH STROKE

In this study , stroke patients with cardiac lesion were 21% . In arabian stroke study [26] cardiac causes were 11%.

In stroke data bank 1986 [27] study cardiac causes were 23%

CVT

Here both CVT cases were puerperial women. Average age was 27. Where as study from Cantu C , barinagarrementeria F et al [28] showed average age was for CVT in puerperial women is 26 years

8 . CONCLUSION

Stroke commonly affecting males than females.

More chance for getting stroke in the age group between 51 – 70 years. Young stroke was also commonly occurring.

Ischemic stroke was more common than hemorrhagic stroke.

Cerebral venous thrombosis occurred in the puerperial women

No cases of subarachnoid hemorrhage in this study.

Hypertension was the predominant risk factor.

DM was also present.

Hypercholesterolemia was associated with in patients with ischemic stroke.

Smoking , alcoholism , and obesity were also important risk factor.

Onset of stroke was sudden in most of the cases.

Stroke developed while on Activities of daily living , then stress

Hemiplegia or hemiparesis was the commonest presentation

Loss of consciousness , Bladder / bowel involvement , UMN type Facial palsy , Aphasia was commonly associated features.

Patients were also presented with TIA symptoms.

Stroke patients had cardiac lesions like IHD , RHD , DCMP , MVP, Aortic sclerosis.

Various medical diseases also were present in stroke patients also.

Stroke caused by Cerebral venous thrombosis commonly occurred in the young females in puerperial period.

In imaging studies ,Cerebral infarction was the common finding. Others were Cortical atrophy, Multiple infarct. Some patients showed normal studies.

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10 . PROFORMA

Name :

Age : **Sex :** **Ip No :**

Address : **occupation :**

Chief complaints :

History of present illness :

Onset

Activity of onset

Associated features

Past History : HT / DM / TIA / PT / BA / COPD / CRF / CAHD / DCMP / RHD / AS / MVP / Hepatic diseases / Seizures / Psychiatric disorders

Personal History : Smoking / Alcoholism / Diet

General examination

Vital signs : pulse rate / blood pressure

Clinical examination

Clinical diagnosis

Investigations :

Urine albumin

sugar

deposits

blood DC / TC / Hb /ESR

blood sugar

urea

creatinine

electrolytes ,

total cholesterol

ECG

Xray Chest

ECHO

Special investigations for associated diseases

CT Brain

plain

contrast (if necessary)

MRI Brain (if necessary)

Final Diagnosis

11 . MASTER CHART

No	Age	Sex	CP	On	Af	Ca	HT	DM	HC	Sm	Al	TIA	CT
1	20	M	RHP	S	FP/A	-	-	-	-	-	-	-	I
2	56	F	FB MD	S	FP	-	+	+	-	-	-	-	I
3	32	F	LHP	G	FP	MVD	-	-	-	-	-	-	N
4	49	M	LHP	S	FP	-	+	-	-	-	-	-	ICH
5	50	M	RHP	S	FP/A	DCMD	+	-	-	-	+	-	I
6	28	F	RHP	G	FP/H	-	-	-	-	-	-	-	CVT
7	47	M	RHP	S	FP/A	-	+	-	-	+	+	-	I
8	84	M	RHP	S	FP/A	IHD	-	+	-	-	-	-	MI
9	72	F	LHP	S	FP	-	+	-	-	-	-	-	CA
10	36	F	LHP	S	FP	-	-	-	-	-	-	+	I
11	67	M	RHP	S	FP/A	-	-	+	-	+	+	-	ICH
12	39	F	RHP	S	FP	RHD	-	-	-	-	-	-	I
13	64	F	RHP	S	FP/A	-	+	-	-	-	-	-	ICH
14	75	M	CS	G		-	+	-	-	-	-	+	CI
15	62	M	RHP	S	FP/A	IHD	+	+	-	-	-	-	CA
16	30	F	TIA	S	FP	-	-	-	-	-	-	-	N
17	57	M	RHP	S	FP/A	-	+	-	+	-	-	-	I
18	82	F	LHP	S	-	-	+	+	-	-	-	-	ICH
19	78	F	RHP	G	FP/A	-	+	+	-	-	-	-	I

No	Age	Sex	CP	On	Af	Ca	HT	DM	HC	Sm	Al	TIA	CT
20	70	M	RHP	S	FP/A	-	+	-	-	-	-	-	I
21	40	F	FBM/P	S	FP	RHD	-	+	+	-	-	-	I
22	27	M	RHP	S	FP/A	-	-	-	-	-	-	-	I
23	50	M	FBM/P	S	FP/A	IHD	+	+	+	+	+	+	I
24	30	M	LHP	S	FP	-	-	-	-	+	+	-	I
25	47	M	TIA	S	FP	-	+	-	-	+	+	+	N
26	52	F	TIA	S	A	IHD	+	-	+	-	-	-	MI
27	54	F	LHP	S	FP	-	+	+	-	-	-	-	ICH
28	60	F	RHP	G	-	-	+	+	+	-	-	+	I
29	60	F	LHP	S	-	-	+	+	-	-	-	-	I
30	57	F	LHP	S	-	-	+	-	-	-	-	-	I
31	48	M	RHP	S	FP/A	-	-	-	-	+	+	+	I
32	51	M	RHP	S	FP/A	DCMP	+	+	-	+	+	-	I
33	60	M	RHP	S	FP/A	-	+	-	-	+	+	-	CA
34	51	M	RHP	S	FP/A	-	+	-	-	+	+	+	CA
35	69	M	LHP	S	FP	-	+	-	-	+	+	-	I
36	64	M	RHP	S	FP/A	-	+	-	+	-	-	-	I
37	55	F	LHP	S	-	-	-	-	-	-	-	-	N
38	42	M	FBM/L	G	FP	-	-	-	-	+	+	-	I

No	Age	Sex	CP	On	Af	Ca	HT	DM	HC	Sm	Al	TIA	CT
39	73	M	RHP	S	FP/A	-	+	-	-	-	-	-	MI
40	26	F	RHP	S	FP/A	-	-	-	-	-	-	-	CVI
41	61	F	RHP	S	A	-	+	+	-	-		-	ICH
42	42	M	TIA	G	-	-	-	-	-	-	-	+	N
43	50	M	RHP	G	FP	-	+	-	-	+	+	-	I
44	52	M	LHP	S	FP	-	-	-	-	-	-	-	MI
45	51	M	LHP	S	FP/A	-	+	-	-	+	+	-	ICH
46	70	M	RHP	G	FP/A	-	+	-	-	-	-	-	CA
47	80	M	RHP	S	FP/A	-	+	-	-	-	-	-	N
48	45	F	LHP	S	FP	MVP	+	-	-	-	-	-	N
49	76	F	RHP	S	-	-	+	-	-	-	-	-	I
50	44	M	FBM/I	S	FP	-	-	-	-	+	+	-	N
51	48	M	FBM/P	G	FP/A	-	+	-	-	+	+	+	I
52	53	M	RHP	S	FP/A	-	+	-	-	+	+	-	ICH
53	52	M	RHP	S	FP/A	-	-	-	-	-	-	-	I
54	55	M	LHP	S	FP	-	-	+	-	-	-	-	ICH
55	63	M	RHP	S	FP/A	-	+	+	-	+	+	-	MI
56	66	M	LHP	S	FP	-	+	-	-	-	-	-	ICH
57	62	F	RHP	G	FP	DCMP	+	-	+	-	-	-	I

No	Age	Sex	CP	On	Af	Ca	HT	DM	HC	Sm	Al	TIA	CT
58	39	M	RHP	G	FP/A	MVP	-	-	-	-	-	-	I
59	40	M	FBM/I	G	FP	-	+	+	+	+	+	-	I
60	59	F	LHP	S	-	-	+	+	-	-	-	-	ICH
61	50	M	LHP	G	-	-	-	-	-	-	-	-	MI
62	58	M	TIA	S	-	-	+	+	-	-	-	-	N
63	64	F	RHP	G	-	-	+	-	-	-	-	-	I
64	49	M	RHP	S	FP/A	-	-	-	-	+	+	-	I
65	54	M	LHP	S	FP	AS	-	-	-	-	-	-	CA
66	54	M	LHP	S	FP	-	-	+	-	-	+	-	N
67	53	M	RHP	G	FP/A	-	+	-	+	+	+	-	I
68	59	M	RHP	S	FP/A	-	+	+	-	+	+	-	CA
69	55	M	RHP	G	FP/A	-	-	+	-	+	+	-	N
70	84	M	RHP	G	FP	DCMP	-	-	-	-	-	-	CA
71	69	F	RHP	S	A	-	+	-	-	-	-	+	I
72	60	F	LHP	S	FP	-	+	-	-	-	-	-	ICH
73	30	M	RHP	S	FP/A	-	-	-	+	-	-	-	I
74	45	M	RHP	S	FP/A	AS	+	+	-	-	-	-	N
75	62	F	RHP	S	FP/A	-	+	+	+	-	-	-	CA
76	74	F	RHP	S	A	-	+	-	-	-	-	-	I

No	Age	Sex	CP	On	Af	Ca	HT	DM	HC	Sm	Al	TIA	CT
77	80	M	RHP	S	FP/A	-	+	-	-	-	-	-	ICH
78	71	M	RHP	G	FP	IHD	+	+	-	-	-	-	I
79	68	M	CS	G		IHD	+	-	-	-	+	-	CI
80	59	F	RHP	S	FP	-	+	-	-	-	-	-	ICH
81	60	M	TIA	S	-	-	+	+	-	+	+	+	N
82	49	M	LHP	S	FP	-	+	-	-	+	+	-	I
83	60	F	RHP	G	FP	-	+	-	-	-	-	-	MI
84	16	M	RHP	S	FP/A	-	-	-	-	-	-	-	I
85	43	M	RHP	S	FP/A	-	-	-	-	+	+	-	I
86	67	F	LHP	G	-	-	+	+	+	-	-	-	MI
87	41	M	LHP	S	FP	-	-	-	-	-	-	-	I
88	32	M	FBM/I	S	FP	-	-	-	-	-	-	-	I
89	55	F	TIA	S	-	-	+	-	-	-	-	-	N
90	46	M	FBM/P	G	FP/A	-	-	-	+	-	-	-	I
91	66	F	LHP	S	-	-	+	-	-	-	-	-	I
92	58	M	RHP	S	FP	-	+	+	-	+	+	-	ICH
93	56	M	LHP	S	FP	-	-	-	-	-	-	-	I
94	58	M	RHP	S	FP/A	DCMP	-	-	-	+	+	-	MI
95	65	M	RHP	G	FP/A	-	+	-	-	+	+	-	CA

No	Age	Sex	CP	On	Af	Ca	HT	DM	HC	Sm	Al	TIA	CT
96	38	M	TIA	G	A	-	-	-	-	-	+	+	N
97	63	F	LHP	S	FP	-	+	+	-	-	-	-	ICH
98	59	F	LHP	G	-	-	+	+	-	+	+	-	CA
99	58	M	RHP	G	FP/A	-	+	-	-	+	+	-	I
100	70	M	RHP	S	FP/A	-	+	+	-	+	+	-	MI
101	66	M	LHP	G	FP	IHD	+	-	+	+	+	+	I
102	81	M	LHP	G	FP	-	+	+	-	-	-	-	CA
103	62	M	RHP	S	FP/A	-	+	-	-	-	-	-	ICH
104	51	M	RHP	G	FP/A	-	-	+	+	+	+	-	I
105	59	M	LHP	G	FP	DCMP	+	+	-	+	+	-	I
106	69	F	LHP	S	FP	-	+	-	-	-	+	-	I
107	80	F	LHP	S	-	-	+	+	-	-	-	-	I
108	55	F	LHP	S	A	-	+	-	-	-	-	-	I
109	28	M	FBM/P	S	FP/A	RHD	-	-	-	-	+	-	I
110	33	M	LHP	S	FP	-	-	-	-	-	-	-	N
111	67	M	LHP	S	FP	IHD	+	-	-	-	+	-	MI
112	65	M	LHP	S	FP	-	+	-	-	+	+	-	ICH
113	64	M	RHP	G	FP/A	-	+	-	-	-	-	+	MI
114	69	M	RHP	S	FP/A	AS	-	-	+	+	+	+	I

No	Age	Sex	CP	On	Af	Ca	HT	DM	HC	Sm	Al	TIA	CT
115	82	M	RHP	S	FP	-	+	-	-	-	-	-	ICH
116	63	M	RHP	G	FP/A	-	+	+	+	+	+		I
117	69	M	RHP	G	FP/A	-	+	+	+	-	-	-	I
118	58	F	RHP	S	FP/A	-	+	+	+	-	-	-	I
119	67	M	LHP	S	FP	-	+	-	-	-	-	-	ICH
120	61	M	LHP	G	FP	-	+	+	-	+	+	-	CA
121	56	M	RHP	S	FP/A	-	+	-	+	-	-	-	I
122	31	M	RHP	S	FP/A	-	-	-	-	-	+	-	I
123	70	F	LHP	G	FP	-	+	-	-	-	-	-	ICH
124	38	F	TIA	S	-	-	+	+	-	-	-	-	N
125	59	F	LHP	S	FP	-	+	-	-	=	=	-	I
126	54	M	RHP	G	FP/A	-	-	-	-	-	-	-	CA
127	60	M	RHP	S	FP/A	-	+	-	-	+	+	-	MI
128	53	M	RHP	G	FP/A	-	+	-	-	+	+	-	ICH
129	69	M	LHP	G	FP	IHD	+	+	+	+	+	+	I
130	68	M	RHP	S	FP	-	+	-	-	-	-	-	ICH
131	78	M	RHP	G	FP	IHD	+	+	+	-	-	-	CA
132	29	F	TIA	S		RHD	-	-	-	-	-	-	N
133	70	F	RHP	S	FP	-	+	-	-	-	+	-	ICH

No	Age	Sex	CP	On	Af	Ca	HT	DM	HC	Sm	Al	TIA	CT
134	40	F	LHP	G	FP	MVP	+	-	-	-	-	-	I
135	70	F	RHP	S	FP	-	+	-	-	-	+	-	ICH
136	78	M	LHP	G	FP	DCMP	+	+	-	-	-	-	CA
137	61	M	RHP	S	FP/A	AS	+	+	-	+	+	-	I
138	68	M	RHP	S	FP/A	-	-	-	+	-	-	-	I
139	60	M	LHP	S	FP	-	+	-	-	+	+	-	ICH
140	82	F	LHP	S	-	-	+	+	-	-	-	+	I
141	44	F	RHP	S	FP	-	+	-	-	-	-	-	N
142	48	F	TIA	S	-	-	+	+	-	-	-	+	N
143	34	M	LHP	S	FP	-	-	-	+	-	-	-	I
144	61	F	RHP	G	FP	-	-	-	+	-	-	-	I
145	44	M	TIA	S		-	-	-	-	+	+	-	N
146	32	M	TIA	S	A	MVP	-	-	-	+	+	+	I
147	58	F	LHP	S	FP	DCMP	+	+	-	-	-	+	CA
148	64	M	RHP	S	FP/A	-	-	-	-	+	+	-	ICH
149	66	M	LHP	S	FP	-	+	-	-	-	-	-	I
150	76	M	RHP	G	FP/A	-	+	-	-	-	-	+	I
151	68	M	LHP	G	FP	-	+	-	-	+	+	-	MI
152	60	M	LHP	G	FP	DCMP	+	-	-	+	+	-	N

No	Age	Sex	CP	On	Af	Ca	HT	DM	HC	Sm	Al	TIA	CT
153	57	M	RHP	S	FP/A	-	+	-	-	+	+	-	ICH
154	59	M	RHP	S	FP/A	-	+	-	-	-	-	-	CA
155	69	F	LHP	S	-	-	+	-	-	-	-	-	CA
156	36	M	RHP	S	FP/A	-	-	-	-	+	+	-	I
157	62	F	RHP	S	FP/A	AS	+	-	-	-	-	-	I
158	37	M	TIA	G	-	-	-	-	-	-	-	-	N
159	58	M	RHP	G	FP/A	-	-	-	-	+	+	-	MI
160	51	M	RHP	S	FP/A	-	-	+	-	-	-	-	ICH
161	57	M	RHP	G	FP/A	-	-	-	-	+	+	-	I
162	53	M	LHP	S	FP/A	-	+	-	-	+	+	-	I
163	66	F	RHP	S	FP/A	-	+	-	-	-	-	-	CA
164	35	M	RHP	G	FP	-	-	-	-	-	-	+	N
165	64	F	RHP	S	-	-	+	-	-	-	-	-	I
166	56	M	RHP	G	FP/A	-	+	+	-	+	+	-	ICH
167	55	M	LHP	S	FP	AS	-	-	-	+	+	+	I
168	65	M	RHP	G	FP	-	+	-	-	+	+	-	CA
169	58	M	LHP	S	FP	-	-	+	-	+	+	-	MI
170	66	M	RHP	G	FP/A	IHD	-	-	+	+	+	-	N
171	53	M	LHP	S	FP	-	+	-	-	+	+	-	ICH

No	Age	Sex	CP	On	Af	Ca	HT	DM	HC	Sm	Al	TIA	CT
172	74	M	RHP	G	FP	-	+	-	-	-	-	-	MI
173	67	M	LHP	S	FP	+	+	-	-	+	+	-	ICH
174	54	M	LHP	G	FP	AS	-	+	-	+	+	-	CA
175	62	F	RHP	S	FP	-	+	-	-	-	-	-	I
176	40	M	LHP	G	FP	MVP	+	+	-	-	-	-	N
177	52	F	RHP	G	FP	-	+	-	-	-	-	+	I
178	53	M	LHP	S	FP	-	+	-	-	+	+	-	ICH
179	72	M	LHP	S	FP	-	+	+	-	-	-	-	ICH
180	64	M	RHP	G	FP/A	-	+	-	-	+	+	-	I
181	62	M	RHP	G	FP/A	-	+	-	-	-	-	-	CA
182	55	M	LHP	G	FP	-	+	-	-	+	+	+	I
183	73	F	TIA	S	-	-	+	-	-	-	-	+	CA
184	35	M	LHP	G	FP	-	-	-	-	+	+	-	N
185	58	M	RHP	S	FP/A	-	-	+	-	+	+	+	ICH
186	53	M	RHP	G	FP/A	-	+	-	+	-	-	-	I
187	66	F	LHP	S	FP	-	+	-	-	-	-	-	ICH
188	38	M	TIA	G	FP	-	+	-	-	-	+	-	N
189	52	F	RHP	S	-	-	+	+	-	-	-	-	N
190	54	M	RHP	S	FP	-	+	-	-	+	+	-	ICH

No	Age	Sex	CP	On	Af	Ca	HT	DM	HC	Sm	Al	TIA	CT
191	54	M	RHP	S	FP/A	-	+	-	-	-	+	+	ICH
192	66	M	LHP	G	FP	-	+	-	-	+	+	-	I
193	62	M	LHP	G	FP/A	-	-	+	-	-	-	-	ICH
194	59	M	LHP	S	FP	DCMP	+	-	-	-	-	-	I
195	60	M	RHP	G	FP/A	-	+	-	-	+	+	-	CA
196	70	M	LHP	S	FP	-	+	+	-	-	-	-	ICH
197	52	M	RHP	S	FP/A	AS	-	-	-	+	+	-	I
198	63	M	RHP	S	FP/A	-	-	-	-	+	+	-	N
199	65	M	RHP	S	FP/A	IHD	-	-	+	-	-	+	CA
200	52	M	RHP	G	FP/A	AS	-	-	-	-	-	-	MI

Serial no / Age / Sex / CP - Clinical presentation (RHP - Right hemiplegia , LHP - Left hemiplegia , FBM - Faciobrachial monoplegia , TIA - Transient ischaemic attack , CS - Cerebellar symptoms) / On – onset (s – sudden , G – gradual) / A.f - Associated features (Fp - Facial palsy , A – Aphasia) / Ca - Cardiac lesions associated with stroke (RHD - Rheumatic heart diseases , IHD - Ischemic heart disease , MVP -Mitral valve prolapse ,DCMP – Dilated Cardiomyopathies , AS - Aortic sclerosis) / HT – hypertension / DM – diabetes / HC - Hyper cholesterolemia / Sm – smoking / Al – alcohol / CT – CT brain (I - Cerebral infarction , ICH - Intra cerebral hemorrhage , N – normal , MI - Multiple infarct CA - Cortical atrophy , CVT - Cerebral venous thrombosis)